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How bad is to be slow-reacting?

On the effect of the delay in response to a changing environment on a population's survival

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Abstract. We consider a simple-model population, whose individuals react with a certain delay to temporal variations of their habitat. We investigate the impact of such a delayed-answer on the survival chances of the population, both in a periodically changing environment, and in the case of an abrupt change of it. It is found that for populations with low degree of mutation-induced variability, being "slow-reacting" decreases the extinction risk due to environmental changes. On the contrary, for populations with high mutation amplitude, the delayed reaction reduces the survival chances.

PACS. 87.23.Cc Population dynamics and ecological pattern formation – 87.18.Tt Noise in biological systems – 87.10.Rt Monte Carlo simulations

1 Introduction

In the present-day context of global warming and habitat destruction, there is an enhanced general interest in the impact of environmental changes on biological populations evolution. Despite this, little has been done from a theoretical point of view, in the frame of evolutionary dynamics modeling, towards a *systematic* approach of the role of various elements involved in these complex circumstances on the population dynamics. In a recent paper [1] we investigated systematically the role of the selection pressure and mutation amplitude, as well as the impact of the quality and quantity of the habitat changes on the behavior of a single-species population.

For simplicity and in order to extract the generic features, we considered the case of a *periodically changing environment*, as in references [2–4]. The case of an abrupt change in the environment was also addressed. The mean-field level of description of the chosen model allowed us to put the finger, for the first time, on the very origin of the emerging complex behavior of this highly-nonlinear system, that is the delicate *interplay between the different time-scale processes*. The role of the amplitude and period of the environmental changes on the critical value of the selection pressure (corresponding to a phase-transition "extinct-alive" of the population) was clarified. However, the intrinsic stochasticity, the dynamically-built correlations between the individuals, and the role of the mutation-induced variety in population's evolution cannot be appropriately accounted for at a mean-field level.

A more refined level of description, which is an individual-based one, was therefore also considered. The main conclusions were that the inherent fluctuations do not destroy the phase transition "extinct-alive", and the mutation amplitude strongly influences the value of the critical selection pressure, giving rise, in particular, to a diversity-induced resonance phenomenon [5,6]. The phase diagram in the plane of the selection and mutation parameters was discussed as a function of the environmental variation characteristics. In particular, an important aspect well-known to experimental biologists, see e.g. [7], was emerging naturally, namely that a small amount of randomness, due to mutations, is beneficial for population's survival in the changing environment, while a too large amount definitely is detrimental to it. The differences between a smooth variation of the environment and an abrupt, catastrophic change were also clarified, pointing to the beneficial role of the mutation in ensuring species survival after a catastrophe.

In this short paper we shall address another aspect of this survival problem, namely the role of the *delay* in the "reactions" of the individuals. The lagged response to environmental changes is a phenomenon widespread in nature [8–13]. However, an extensive theoretical analysis of its impact on population dynamics is still lacking. The role and effects of time-delay in biological systems has been addressed previously in the context of Lotka-Volterra type of dynamics of interacting species [14], where the "delay" was included at the level of the coupling between the species.

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Here we are considering a different problem, namely the delayed-response of the individuals of a single-species population to a changing environment. Using a simple model, we shall try to clarify the degree and limits of validity of the commonly-spread belief that "a population of fast-reacting individuals has better survival chances face to changes in their environment".

2 Model

We consider the same type of model as in reference [1], namely a population of *hermaphrodite* individuals (i.e., which, although bisexual, need mating for reproduction), living on a two-dimensional square lattice of size $L \times L$. We assume that the individuals cannot cross the borders of the lattice. Moreover, the lattice has a *finite carrying capacity*, which comes from the exclusion assumption that there is at most one individual in each lattice node.

The dynamics of the population takes place at discrete time-steps and is the result of: natural selection (interaction with the environment), individual motion, mating and reproduction, as described below.

A. Natural selection. Individual trait, time-dependent optimum, fitness, delayed-response, selection pressure, extinction probability

Each individual i is characterized by its *trait* or *phenotype*, which for simplicity is represented here through a real number $z_i \in [0,1]$. The trait is fixed once and for all at the birth of the individual.

The population lives in an environment whose influence on the individuals is encoded in the value of the so-called *optimum*, $\varphi \in [0,1]$, which we suppose to be homogeneous in space, but *periodically variable in time* $\varphi = \varphi(t)$. Moreover, we consider here the simplest possibility,

$$\varphi(t) = 0.5 + A \sin\left(2\pi \, \frac{t - t_{\rm init}}{T_0}\right) \Theta(t - t_{\rm init}) \,. \quad (1)$$

Here A denotes the amplitude of the environmental oscillation, with $0 < A \leqslant 0.5$, T_0 is its period, and $t_{\rm init}$ is the moment of onset of the optimum perturbation; Θ is the Heaviside step-function.

The case of an abrupt change in the environment, for which the optimum jumps at $t = t_{\rm init}$ from $\varphi = 0.5$ to $\varphi = 0.5 + A$ was also considered.

An individual i "reacts" with a certain specific delay τ_i to the changes in the environment. This means that its instantaneous *fitness* (or "adequacy to the environment", see below) at time t is determined by the value of the optimum at a previous time $(t - \tau_i)$,

$$f_i(t) = 1 - |z_i - \varphi(t - \tau_i)| \,. \quad (2)$$

The fitness determines the *instantaneous individual extinction probability per time step* $p_i(t)$, according to the following expression:

$$p_i(t) \, = 1 - \, \exp\left[-\frac{\mathcal{S}}{f_i(t)}\right] \,, \quad (3)$$

where $\mathcal{S}$ is a parameter which models the *selection pressure* of the environment and constitutes a main control parameter of the system. During its life-time, an individual oscillates cyclically from being perfectly-adapted, when $z_i = \varphi(t - \tau_i)$, i.e., from a minimum possible extinction rate $p_i(t) = \exp(-\mathcal{S})$, to a worse adaptation, which corresponds to $z_i \neq \varphi(t - \tau_i)$ and to a larger instantaneous extinction probability, and finally to a total lack of adaptation, when $p_i(t) = 1$. The pool of adapted individuals changes thus at each time step.

The choice (3) we made of the extinction probability and the implicit definition of the selection pressure parameter $\mathcal{S}$ are frequently encountered in the biological literature, see e.g. [15]. Other choices and thus other ways of measuring the "selection pressure" are of course possible. However, most of them can be mapped one onto the other and/or account for equivalent *qualitative* aspects of the interaction between the individuals and their environment.

The *individual delayed-response time* τ_i is fixed once and for all at one individual's birth. We consider here a simple case when the τ_i's are random variables drawn from an uniform distribution within an interval $[0,\, T_d]$. The upper limit of this interval T_d represents another control parameter of the model. It is obvious that for a periodic variation of the environment only the values $T_d < 2T_0$ are relevant.

Note also that an equal delay-time for all individuals amounts simply to a change in the time-origin. As such, a mean-field level description of the population dynamics (which is already known as inappropriate for describing mutation, see [1]) will not be able to account for the effects of the individual delay-times on the global evolution of the population. We shall therefore focus exclusively on the individual-based numerical simulations.

B. Individual motion

An individual can move to its surroundings, and the simplest possibility that we shall adopt hereafter is a random-walk. Namely, in one time step the individual jumps on the lattice, from its initial location to a randomly chosen nearest-neighbor one (i.e., a site within the von Neumann neighborhood of the initial node), provided that the chosen site is empty, and that it lies within the boundaries of the system. If none of the four first-neighbor nodes is empty, then the individual cannot move, and thus cannot mate (see below).

C. Mating and reproduction. Heredity and mutation

Suppose an individual i reaches a destination node. If there are other individuals ("neighbors") in the nearest-neighborhood of this destination site, then the individual "i" chooses at random one of these neighbors, call it "j", for mating. The pair of individuals i and j may then give birth to as many offsprings as there are empty nodes in their surrounding joint nearest neighborhoods. Therefore, all the empty nodes closely surrounding the two parents will be filled with offsprings. The maximum number of offsprings of one pair is thus equal to six (corresponding to a completely empty nearest neighborhood of the parents).

The trait of a progeny k coming from parents i and j is determined by the parents' traits (heredity), but it can

also present some "variations" due to different random factors, such as recombination, mutations, etc. We shall assume that

$$z_k = \frac{1}{2}(z_i + z_j) + m_k \,, \tag{4}$$

where m_k represents these variations. It brings diversification into the phenotypic pool of the population and we call it conventionally *mutation*. For simplicity, we shall admit that m_k is a random number, uniformly distributed in the interval $[-\mathcal{M}, \mathcal{M}]$, where $0 < \mathcal{M} < 1$ is called hereafter the *mutation amplitude* and is a control parameter of the system[1]. Moreover, if equation (4) leads to $z_k > 1$ or $z_k < 0$, then one "renormalizes" it by resetting z_k to 1, respectively 0, which means simply that the trait of the individuals cannot overcome some fixed limits. This choice (4) for the trait of an offspring is often made in the biological literature [15].

The population dynamics is thus driven by two main "forces" that are acting, to some extent, in opposite directions: selection and mutation, characterized, respectively, through the values of the control parameters $\mathcal{S}$ and $\mathcal{M}$. Selection, combined with heredity, tries to bring the average trait close to the optimum, while mutation introduces diversity in the individual traits, and thus is broadening the distribution of the population's traits.

The Monte-Carlo simulation algorithm considers the individuals distributed on the lattice nodes, the initial condition being represented by their positions, the prescribed values of the individual traits and delay-times. The initial $N(0)$ individuals are randomly-distributed with a mean concentration $c(0) = N(0)/L^2$, and their individual traits are randomly assigned from an uniform distribution between 0 and 1.

The individuals are evolving, at discrete Monte-Carlo time steps (MCS, defined hereafter), according to the stages A–C of the dynamics as described above, namely:

A. At a given time t an individual i is picked at random, and its extinction probability $p_i(t)$, corresponding to one MCS, is determined according to equations (2, 3). Then a random number r is extracted from an uniform distribution in the range [0, 1]; if $r < p_i$, the individual dies, otherwise it survives.

B. If it survives, the individual i jumps at random to one of the empty nearest-neighbor nodes on the lattice.

C. Then it possibly mates and produces offsprings.

If at the time t there are $N(t)$ individuals in the system, then the above steps A–C are repeated $N(t)$ times; this constitutes one MCS, the unit-time of the simulations. Afterwards, the time is advanced by one step, $t \to t + 1$, and the above algorithm is repeated.

As a last remark on the model, it is known on general backgrounds [16] that the system size is playing a certain role on the location of the phase transition point, as well as on its "sharpness". We used for all our Monte Carlo simulations a system of 100×100 lattice sites, for which we had shown previously, see reference [1], that the qualitative features of the phase diagram are practically not affected by finite-size effects.

[1] In the biological literature parameters analogous to $\mathcal{M}$ are often referred to as *mutation rate*. However, because of the physical aspect $\mathcal{M}$ designates, the term *mutation amplitude* looks more appropriate to us.

3 Results

For a periodic oscillation of the optimum we investigate the temporal evolution of a population starting from a given initial concentration $c(0)$. Depending on the characteristic parameters, the population can evolve, on the average, either to an "alive phase", for which its concentration is actually oscillating periodically, with period $T_0/2$, around a nonzero mean value, or can get extinct after a transient period of time. In our previous paper [1] we investigated in detail the phase diagram "extinct–alive" of the population in the plane of the control parameters $\mathcal{S}$ and $\mathcal{M}$, for different values of the characteristics A and T_0 of the optimum oscillation. The same type of phase diagram was also constructed for the case of an abrupt jump of the the optimum.

Our principal concern in this paper is to determine how the delay in the individual response to the changing environment – i.e., the value of the control parameter T_d – affects the phase diagram extinct-alive of the system. We performed extensive simulations for various range of parameters and the main results are illustrated in Figure 1.

One notices several interesting features exhibited by these figures:

(a) Consider first the "intermediate" values of T_0 for which, as described in reference [1] for the no-delay case, one encounters the diversity-induced resonance phenomenon, i.e., the "peak" in the phase-diagram illustrated in the upper and middle panels of Figure 1. Then:

(i) For small values of the mutation amplitude $\mathcal{M}$, the existence of a delay in the response of the individuals to environmental changes (i.e., $T_d \neq 0$) is *increasing the survival chances* of the population. The diversity related to the randomness in the response of the individuals can contribute to the appearance of a larger pool of well-adapted individuals and is thus formally equivalent to an increase in the "effective" mutation amplitude, which is beneficial for the survival [1].

(ii) For large values of $\mathcal{M}$, however, adding the randomness of the delayed-response to the mutation-related one is leading to an even higher "effective" mutation amplitude. As such, the extinction risk of the population is increased: as seen in the plots, the phase diagram for the populations with delayed-response ($T_d \neq 0$) lies always below the one of the instantaneously-reacting population ($T_d = 0$).

(iii) Finally, the peak related to the mutation-induced diversity is generally still present for the systems with time delay. However, in this case the randomness in the delayed-response can turn a part of the pool of well-adapted individuals into less-adapted ones, and thus the height of the peak is reduced as compared to the case of an instantaneously-adapting population.

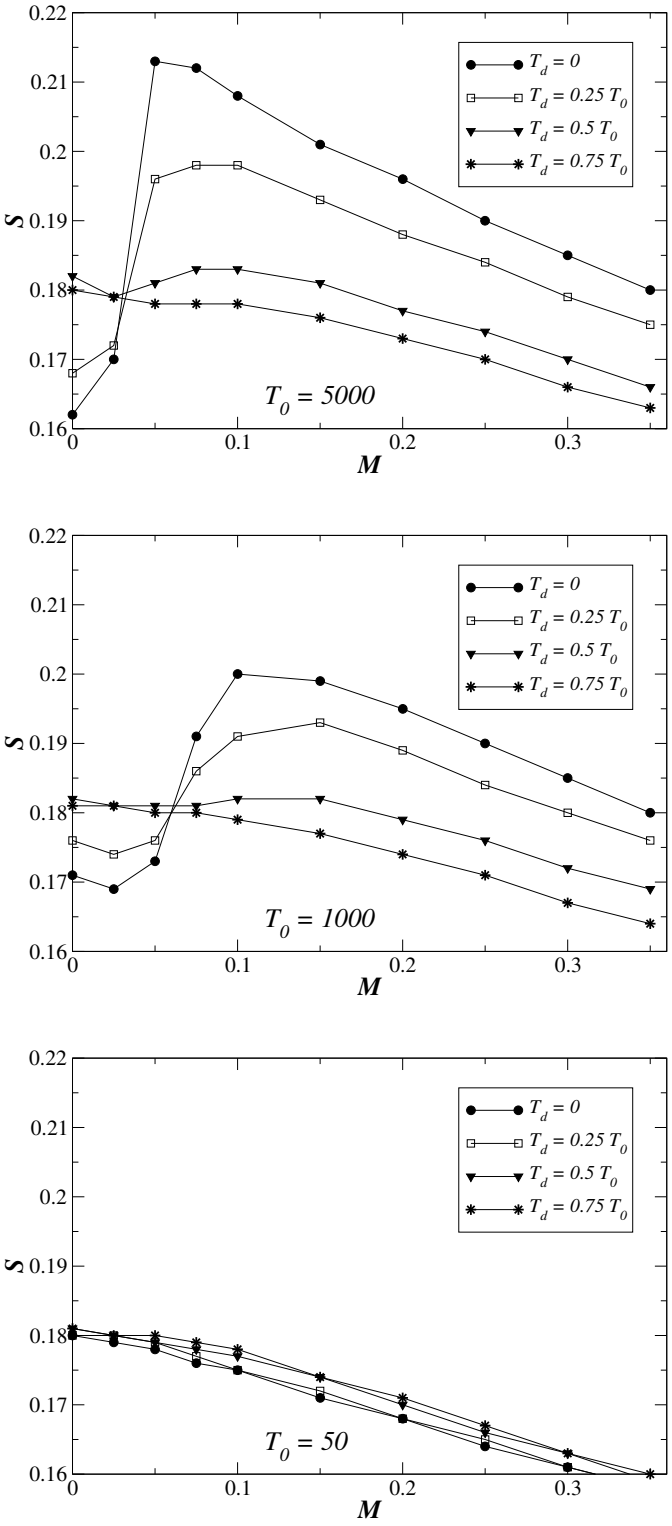

Fig. 1. Phase diagram extinct (above the curve) – alive (below the curve) in the plane of the selection pressure $\mathcal{S}$ and mutation amplitude $\mathcal{M}$, for different values of the delay time T_d and of the optimum oscillation period T_0. From the upper to the lower panel, $T_0 = 5000, 1000$, and 50, respectively; the values of the other parameters are $L = 100$, $c(0) = 0.7$, $A = 0.3$, and $t_{\rm init} = 1000$. The average was taken over 10 realizations of the stochastic dynamics and the estimated errors in the value of the critical selection pressure are less than ± 0.002.

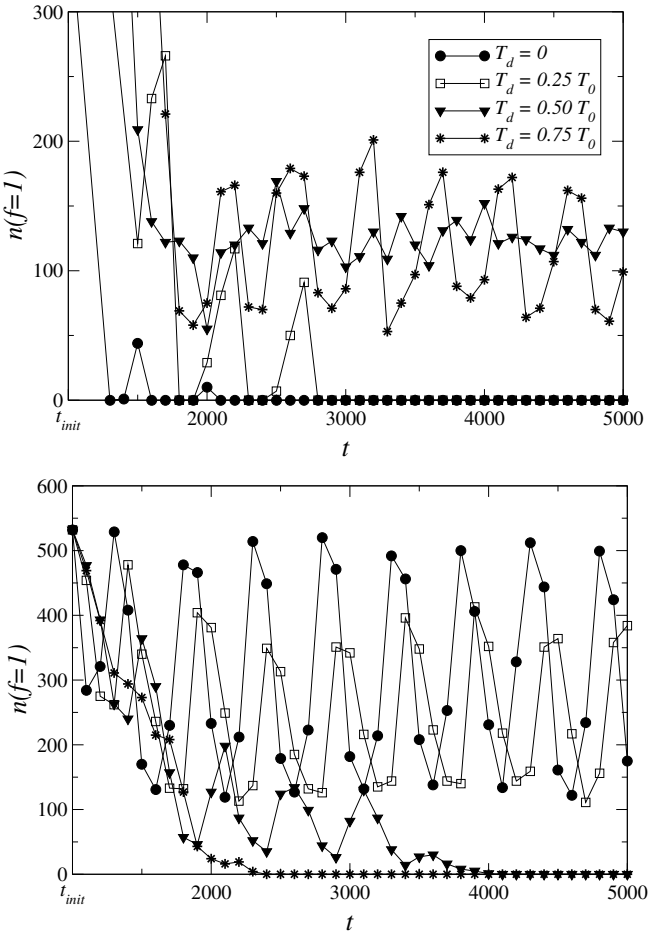

Fig. 2. Time evolution of the number $n(f = 1)$ of the fittest individuals of a population for different values of the delay time T_d. Upper panel: $\mathcal{M} = 0.025$ (small mutation amplitude), lower panel: $\mathcal{M} = 0.1$ (intermediate mutation amplitude). The legend in the upper panel also applies to the lower panel. The values of the other parameters are $L = 100$, $c(0) = 0.7$, $A = 0.3$, $T_0 = 1000$, and $t_{\rm init} = 1000$, corresponding to the middle panel of Figure 1.

For large delays (like $T_d = 0.75\,T_0$ in the figures) this peak can be even suppressed.

(b) One concludes therefore that the role of the delay-induced diversity is an increase in the "effective" mutation amplitude. As such, it can be easily predicted that for small values of T_0 (rapid oscillations of the environment) the dynamics of the system will be only slightly affected by the delay, since it is already only slightly sensitive to changes in $\mathcal{M}$. This is illustrated in the lower panel of Figure 1. No diversity-induced peak, i.e., no optimal "effective" mutation amplitude is encountered in these cases, any mutation and any delay in response being harmful for the survival of the population.

A way to get a better insight into the reasons for this behavior is the monitoring of the temporal evolution of the pool of fittest individuals (i.e., the individuals with $f_i(t) = 1$). Figure 2 illustrates this point, for a fixed value of T_0 and three values of $T_d \neq 0$, corresponding to the

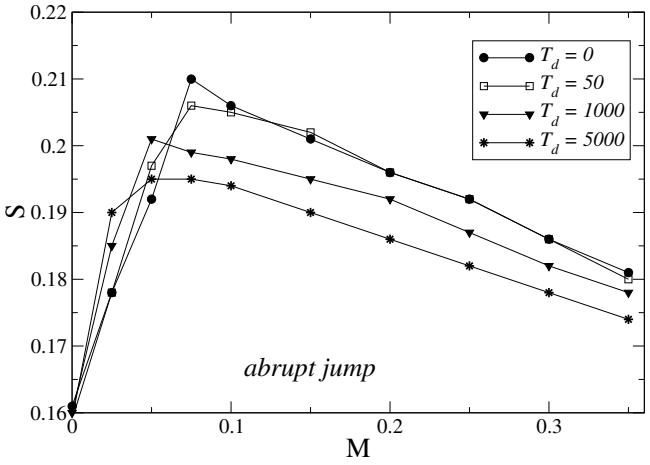

Fig. 3. The phase diagram extinct (above the curve) – alive (below the curve) in the plane of the selection pressure $\mathcal{S}$ and mutation amplitude $\mathcal{M}$, for an abrupt jump in the value of the optimum, from $\varphi = 0.5$ to $\varphi = 0.8$, for different values of the delay time T_d. The values of the other parameters are $L = 100$, $c(0) = 0.7$, $t_{\rm init} = 1000$. The average was taken over 10 realizations, and the estimated errors in the value of the critical selection pressure are less than ± 0.002.

middle panel of Figure 1. The upper panel of Figure 2 pertains to the region of small mutation amplitudes in the phase diagram, for which a delayed-response enhances the survival chances. The lower panel refers to the region of the peak in the phase diagram, for which delay increases the extinction risk.

One can see that for the surviving populations the number $n(f = 1)$ of the instantaneously fittest individuals is oscillating periodically in time (but never reaching zero), while it decays (with oscillations) to zero for the populations that will get extinct. The pool of the fittest individuals is enhanced by the delay-induced diversity in systems with small mutation amplitude (upper panel of Fig. 2) and, on the contrary, it is depleted by the delayed-response in populations with intermediate and large mutation amplitude (lower panel of Fig. 2).

Finally, we addressed also the effects of a delayed answer in the case of a catastrophic, abrupt change in the environment. As illustrated in Figure 3, one encounters the same type of phenomena as in the case of a smooth variation of the optimum, namely the fact that for small mutation rate the largest the delay parameter T_d, the bigger the survival chances of the population.

In order to understand the mechanism underlying this behavior of the populations with small mutation amplitude $\mathcal{M}$, it is useful to follow the temporal evolution of the fitness histogram "number of individuals $n(f)$ versus fitness f". This is done in Figure 4 for two populations that differ only through the value of the delay parameter T_d, such that one of them gets extinct, while the other one survives after the catastrophe. Before the catastrophe, the histogram had an important peak at $f = 1$, and a tail (due to mutations) at low-fitness values. After the catastrophe, a new peak of low-fitted individuals appears, such that

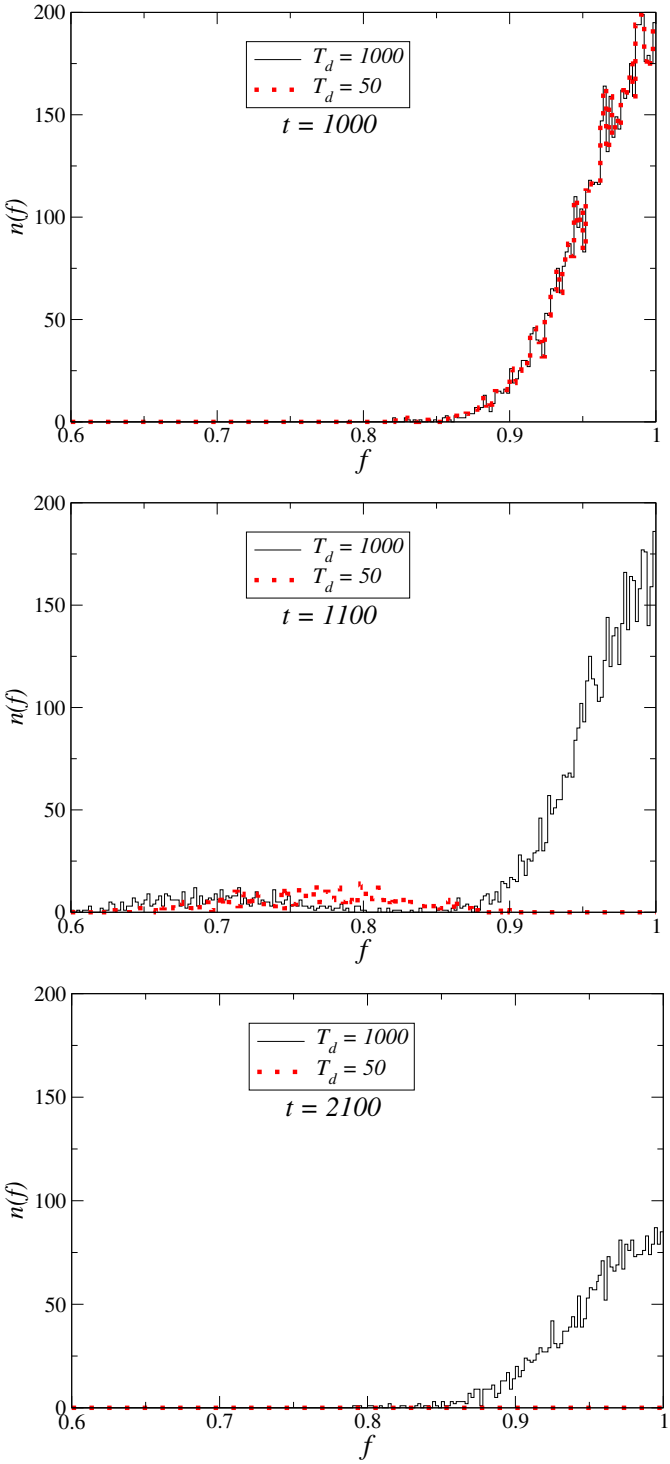

Fig. 4. Fitness histograms: number of individuals $n(f)$ versus fitness f for two populations with low mutation amplitude $\mathcal{M} = 0.05$ and different delay-response parameters $T_d = 1000$ (continuous line) and $T_d = 50$ (dotted line) in case of a catastrophic event. The optimum jumps at $t_{\rm init} = 1000$, from $\varphi = 0.5$ to $\varphi = 0.8$. The upper panel corresponds to time $t = 1000$, just before the optimum jump. The middle panel corresponds to $t = 1100$, and the lower panel to $t = 2100$ (when only the population with $T_d = 1000$ survived). The other parameters are $L = 100$ and $c(0) = 0.7$. Single runs were considered.

the histogram becomes bimodal. One notices that the existence of a larger delay time ensures the persistence of a sufficiently large pool of high-fitted individuals even after the catastrophe, and this pool will ensure the survival of the species till the new-born individuals get adapted slowly, through small mutations, to the new environment. For a surviving population the histogram becomes peaked again, in the long run, around $f = 1$. A shorter delay time T_d, however, cannot ensure this persistence of the high-fitted individuals pool for a long enough time, and the population dies, since the adaptation through mutations is not rapid enough.

As seen in Figure 3, on the contrary, for large mutation amplitudes the larger the delay T_d, the higher the extinction risk, since, as in the case of a periodically-varying environment, in this case the delay-induced stochasticity adds up to the mutation, leading to an even higher effective mutation amplitude, which is harmful for the system.

4 Conclusions

We considered a simple model of single-species population dynamics in a changing environment and we investigated the role of a delayed answer of the individuals to these habitat changes. In the case of a smooth variation of the environment, it was found that, in general, for populations with small mutation amplitudes it is more beneficial, in terms of the survival chance, to be slow-reacting than to answer instantaneously to the variations of the environment. However, for intermediate and large mutation amplitudes, faster reactions are preferable to slower ones. In case of a very-rapidly oscillating environment, the rapidity of reaction influences only slightly the survival chances. The same type of statements holds true for the case of a catastrophic, abrupt jump in the optimum.

As such, one has to be rather cautious with "common-sense" statements of the kind "a population of fast-reacting individuals has better survival chances face to changes in their environment". Of course, more complex and realistic models than the one we presented here are needed in order to make more detailed *quantitative* statements and reliable predictions for real biological systems, and to investigate further aspects of the intricate problem of a population evolving in a changing environment.

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